

The CENTRAL AFRICAN JOURNAL OF MEDICINE

Dr. DAVID LIVINGSTONE

Vol. 4. No. 7.

JULY, 1958.

C O N T E N T S

ORIGINAL ARTICLES

Rift Valley Fever in Rhodesia
Rift Valley Fever in Southern Rhodesia
Bilharziasis in Childhood

Neurosurgical Control of Pain
Pitfalls in Entertaining the Young
Torsion of a Wandering Spleen
Introduction to Medical Writing

L. Stern - - - - - 281
D. K. Shone - - - - - 284
F. Morley Smith and M. Gelfand - - - - - 287
L. F. Levy - - - - - 289
E. C. S. - - - - - 297
W. Shepherd Wilson - - - 299
R. Whitehead - - - - - 301

EDITORIALS

Pseudotumour Cerebri
Hepatic Coma
Presentation to Lord Malvern
Retirement of William Francis Wynne

305
305
306
306

The Queen's Birthday Honours - - - 308
Opening of Lynbrook, Que Que - - - 308
Salisbury Cardiac Society - - - 308
Obituary — Jacob Samuel Liptz - - - 309
In Rhodesia Then - - - - - 310

Correspondence - - - - - 311
Institute on Alcoholism - - - - - 312
Book Reviews - - - - - 313
The Journal Library - - - - - 314
Latest Pharmaceuticals - - - - - 316

PUBLISHED MONTHLY, ANNUAL SUBSCRIPTION £2 2s. 0d.

Registered at the General Post Office as a Newspaper.

Rift Valley Fever in Southern Rhodesia

BY

D. K. SHONE, B.V.SC.

Veterinary Research Laboratory, Salisbury.

Rift Valley fever is a virus disease, characterised by a short incubation period, typical liver lesions and heavy mortality in young lambs and calves. The aetiological agent is an arthropod-borne pantropic virus exhibiting marked hepatotropism. It is chiefly a disease of ruminants, but man and monkeys are highly susceptible, as also several rodent species.

Daubney and Hudson (1931) first recognised the disease as an entity, and as a result of their investigations concluded it was a filterable virus and probably mosquito transmitted. It was not, however, until Smithburn, Haddow and Gillett (1948) isolated the virus from wild caught mosquitoes in the Semliki Forest of Western Uganda that the mode of transmission was definitely established.

Apart from Kenya and Uganda, the virus has been isolated from the Union of South Africa during the course of an outbreak of heavy mortality amongst sheep and cattle in the Western Free State, Southern and Western Transvaal and North-Western Cape Province (Alexander and Dickson, 1951).

Virus neutralising antibodies have also demonstrated the presence of the disease in Uganda, French Sudan, Anglo-Egyptian Sudan and French Equatorial Africa (Findlay, Stefanopoulou and MacCallum, 1936), the Knysna area of the Union of South Africa (Kaschula, 1953), as well as from the Ngamiland and Chobi areas of the Bechuanaland Protectorate (the Director of Medical Services, Bechuanaland Protectorate, 1955).

MATERIALS AND METHODS

1. *Virus Isolation.*—All isolations were obtained in day-old Swiss albino mice by the intracerebral and intraperitoneal routes from specimens forwarded to the Veterinary Research Laboratory, Salisbury. Twenty per cent. emulsions of tissues, lightly centrifuged, were employed. The mice were observed daily over a period of twelve days and the brains of dead and/or sick mice were harvested and stored at 4° C. as a 10 per cent. emulsion in nutrient broth. This brain material was utilised as the source of antigen for the serum-virus neutralisation and pathogenicity tests.

(a) Bovine foetal strains: Eight strains were isolated from liver and/or brain emulsions from aborted bovine foetal specimens, originating from Salisbury, Gatooma, Eiffel Flats and Hartley areas. No preservative was used for any of the specimens. Those from Gatooma and Eiffel Flats were despatched by rail—an overnight journey. The B. & M. strain originating from the Gatooma area has been stored under dry ice refrigeration.

(b) Sheep strain: This strain was isolated from a lamb liver, preserved in 50 per cent. glycerine-saline, submitted during the course of heavy mortality in sheep in the Gatooma area.

(c) Human case: Virus was re-isolated from serum collected from a case of accidental laboratory infection during the period between two febrile reactions.

All strains isolated were subjected to serum-virus neutralisation tests and their pathogenicity for adult mice determined.

2. Antibody Determinations

(1) Quantitative test: A quantitative test was undertaken on acute and post-phase serum collected from a suspect human case L. Equal volumes of serum and tenfold dilutions of virus (B. & M. strain) were mixed and incubated at 37° C. for 60 minutes. A family of day-old Swiss albino mice was utilised for each serum-virus mixture, of which 0.1 ml. was inoculated intraperitoneally.

(2) Qualitative tests: Qualitative tests were undertaken on bovine serum samples submitted from various areas of the country. The P.R.V.F.M. strain (Kaschula, 1953) of Rift Valley fever virus diluted to contain 20,000MLD per millilitre was utilised as the antigen. Equal volumes of the serum under test and the virus solution were incubated at 37° C. for 60 minutes: 0.1 ml. of this mixture was inoculated by the intraperitoneal route into each of a family of day-old mice. Adequate controls were employed in all cases.

RESULTS

Isolation of the Virus.—In the course of examination of specimens, during the period 1st January, 1957, to 30th June, 1957, for viruses pathogenic to infant mice, Rift Valley fever virus was isolated on ten occasions. Eight strains were isolated from aborted foetal specimens; one strain was from a lamb liver, and a re-isolation of virus was made following an accidental laboratory infection.

In Table I are detailed the date of isolation and the type and origin of each specimen. It will be noted that all isolations were made during the period 13th March, 1957, to 1st June, 1957.

The three bovine foetuses from the Salisbury area originated from different farms. Virus was isolated from liver and brain emulsions from two of the foetuses. In the third case no brain material was available and isolation from the liver only was made.

Rift Valley fever virus was isolated from two foetal specimens received from a farm in the Eiffel Flats area and two from a farm in the

Table I
THE ISOLATION OF RIFT VALLEY FEVER
VIRUS STRAINS

Date	Specimen	Origin
13/3/57	Bovine foetus	Salisbury
26/3/57	Bovine foetus	Salisbury
30/3/57	Lamb foetus	Gatooma
6/4/57	Bovine foetus	Eiffel Flats*
18/4/57	Bovine foetus	Gatooma†
30/4/57	Bovine foetus	Eiffel Flats*
30/5/57	Bovine foetus	Salisbury
30/5/57	Bovine foetus	Gatooma†
31/5/57	Human serum	Salisbury
1/6/57	Bovine foetus	Hartley

* The same farm.

† The same farm.

Gatooma area. Two of these specimens consisted of the head only, one of the other specimens consisted of the head and portion of the liver, while the fourth foetal specimen was complete. In the latter two cases virus was isolated from the brain emulsion, but not from the liver emulsion by either the intracerebral or intraperitoneal routes in day-old mice.

The eighth foetal specimen from which virus was isolated was a complete specimen from the Hartley area. Isolations were made from both liver and brain emulsions.

The incubation periods of the original isolations varied from 24 to 48 hours. In the case of the intracerebral route, the period was several hours shorter than by the intraperitoneal route.

Enquiries were made from the farmers as to the state of the dams which aborted and, except for one case where the cow contracted lumpy skin disease shortly after aborting, no clinical symptoms beyond the abortions were noted. There were no apparent indications of a febrile reaction, and two of the cows being milked showed an increase in production following the abortion without any prior drop having been recorded.

In the case of the accidental laboratory infection, it is possible to determine the exact incubation period. Four days after the only occasion on which virulent material was handled by this officer, he first complained of influenza-like symptoms. Gloves were worn when handling the material. A typical diphasic febrile reaction

was recorded. Rift Valley fever virus was isolated from serum collected during the period between the two febrile reactions.

Quantitative Serum Tests.—The incubation period in the veterinary surgeon L., who contracted the disease after conducting an autopsy on a sheep, was also four days. In this case diagnosis was made by comparing the virus neutralising properties of acute and post-phase serum. Acute phase serum was collected on the fourth day after symptoms were first noted and the post-phase serum 68 days later.

Table II
THE VIRUS NEUTRALISING PROPERTIES OF ACUTE
AND POST-PHASE SERUM COLLECTED FROM A
HUMAN CASE CHECKED IN INFANT MICE

Antigen	Virus Dilution	Serum	Results
B. & M. Strain	10 ⁻¹	Acute phase	6/0*
	10 ⁻²		6/0
	10 ⁻³		6/0
	10 ⁻⁴		2/7
B. & M. Strain	10 ⁻¹	Post phase	0/6
	10 ⁻²		0/6
	10 ⁻³		0/8*
	10 ⁻⁴		0/10

* Number of mice dead/number alive at end of twelve days.

From Table II it is seen that the post-phase serum protected against more than three logs of virus as compared with the acute phase serum, and confirmation of infection with Rift Valley fever virus was obtained.

Qualitative Serum Tests

Table III
THE PRESENCE OF NEUTRALISING ANTIBODIES IN
SERA ORIGINATING FROM VARIOUS AREAS
DURING 1955-1956

Area	Total Number Tested	Number Positive
Bulawayo	115	44
Sabi Valley	237	15
Norton	21	7
Nyamandhlovu	10	2
Salisbury	91*	1

* Sixty-six of these samples were from one farm.

From Table III it is seen that the highest number of positive sera was from the Bulawayo area. This is probably due to the wave of abortions recorded in that area in early 1955, being the result of a Rift Valley fever epidemic. The high percentage of positives recorded is probably not a true reflection of the extent of infection in the herds, as cows with a history of a previous abortion were selected.

Neutralising antibodies have also been recorded from the Umtali area.

Very limited numbers of sera submitted from other areas have also been examined, but no neutralising antibodies were demonstrated. Due to extremely limited numbers, the negative results are not of significance.

DISCUSSION

The isolation of Rift Valley fever virus and the demonstration of neutralising antibodies have shown that the virus occurs in a large portion of the country. A more extensive survey would, in all probability, reveal its presence in the major portion of the country, as the climate generally is suitable for the existence of the insect vector.

The isolation of the virus from a number of aborted bovine foetal specimens has established the importance of the disease as an aetiological agent of bovine abortions in this country.

The diagnosis of the disease in humans indicates that it must be considered as an aetiological agent, where influenza-like disease is encountered in those people closely associated with the stock industry.

In two cases virus was isolated from foetal brain tissue, but not liver tissue. This may be due to the early onset of decomposition of the liver as compared to the brain, causing the destruction of the virus in the liver. Brain tissue is for this reason preferable as a source of material for virus isolation, particularly in those cases where decomposition has already become established.

SUMMARY

(1) The isolation of Rift Valley fever virus in Southern Rhodesia for the first time is recorded.

(2) The importance of the virus as an agent of bovine abortion in this country has been established.

(3) Foetal brain tissue is considered to be preferable as a source of material for virus isolation as compared to liver tissue.

(4) The diagnosis of two human cases is recorded.

REFERENCES

- ALEXANDER, R. A. & DICKSON, J. (1951). Rift Valley fever in the Union. *J. S. Afr. Vet. med. Assoc.*, 22, 105-112.
- DAUBNEY, R., HUDSON, J. R. & GARNHAM, P. C. (1931). *J. Path. and Bact.*, 34, 545-579.
- Director of Medical Services, Bechuanaland Protectorate Government, Annual Medical and Sanitary Report for the year 1955, p. 9.
- FINDLAY, G. M., STEFANOPOULOU, G. M. & MACCALLUM, F. O. (1936). *Bull. Soc. Path. Exot.*, 29, 986. Cited by Henning, M. W. (1956). *Animal Diseases of South Africa* (Central News Agency).
- KASCHULA, V. R. (1953). The propagation and modification of strains of Rift Valley fever viruses in embryonated eggs and their use as immunising agents for domestic ruminants. Thesis, University of Pretoria.
- SMITHEBURN, K. C., HADDOW, A. J. & GILLET, J. D. (1948). *Brit. J. Exp. Path.*, 29, 107. Quoted from *Vet. Bull.* (1950), 20, 524.

Acknowledgments

Mr. G. J. Christie, Acting Assistant Director of Veterinary Services (Research), is thanked for facilities and permission to publish this report. The assistance of Dr. R. A. Alexander, Director of Veterinary Services, Onderstepoort, for the examination of a large number of sera for antibodies, is appreciated. Mr. J. Becks is thanked for his technical assistance.



This work is licensed under a
Creative Commons
Attribution – NonCommercial - NoDerivs 3.0 License.

To view a copy of the license please see:
<http://creativecommons.org/licenses/by-nc-nd/3.0/>

This is a download from the BLDS Digital Library on OpenDocs
<http://opendocs.ids.ac.uk/opendocs/>